the strength of the upper, is a topic worthy of consideration, and experience has enabled the writer to commend it most warmly to the attention of his professional brethren.

ART. II.—*A Case of Lateral Sclerosis of the Spinal Cord.* By
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THE time has almost come when it is no longer necessary to plead for a more careful study of nervous diseases even on the part of the busy general practitioner. Our knowledge, both pathological and clinical, of this department of medicine has gained in recent years so enormously, both in extent and in exactness, that it is no longer possible for anyone to neglect a branch of medical science abounding in interest and affording an unrivalled field for the exercise of those powers of observation which are the most striking characteristic of the skilled physician. Yet there are some who still deprecate the expenditure of much time and labour in the investigation of nervous diseases, and apparently on two grounds—first, that the diagnosis of such disease is often obscure and difficult; and secondly, that even if a correct diagnosis be established, treatment is of little or no avail. The first argument had better be given up by those who would shun the imputation of laziness and ignorance. The diagnosis of nervous disease, no doubt, presents very special and peculiar difficulties, but it is being pursued, both in this country and on the Continent, with extraordinary zeal and with distinguished success. Take, for example, such a disease as posterior sclerosis or locomotor ataxia. The last generation of observers scarcely recognised its existence; now our knowledge of the marvellous assemblage of symptoms presented by this malady is minute in its exactness, and far exceeds that which we possess of many familiar diseases which have been under observation from time immemorial.

The second argument—that the treatment of nervous disease is disheartening—is one of those arguments which prove too much. If our clinical studies are to be confined to those diseases in which treatment is strikingly and unquestionably efficacious, the range of our observations is likely to be somewhat limited. Granting to the fullest extent that it is in the realm of nervous disease that therapeutics exhibits its most conspicuous failures, such a fact is no sufficient excuse for the neglect of this department of study—

first, because as our knowledge of pathology and symptomatology grows more perfect we may hope that our treatment will become more successful; and secondly, because therapeutics is not the sole end of diagnosis. The importance of a correct prognosis cannot in many cases be over-estimated, and it is highly characteristic of nervous diseases, that while treatment is purely empirical and so far unsuccessful, the prognosis in most cases admits of a high degree of certainty.

In no department of nervous disease has such progress been made within the last decade as in diseases of the spinal cord. Until recently these affections, so multiform in their symptoms, so pregnant with signs of vital import regarding the life and happiness of the individual, were imperfectly recognised and frequently confused with each other; but order has begun to evolve from this chaos, and the great results already achieved are full of hopeful augury for the future.

It is to one of these diseases that I propose to direct attention; and I shall first relate, as briefly as possible, the clinical history of the case:—

CASE.—James M. was admitted to the Royal Hospital, Belfast, on April 21st, 1883. He was suffering from almost complete loss of power over the lower extremities, with marked rigidity of the muscles. There was some weakness of the upper extremities, with wasting, but in other respects he felt perfectly well. The history elicited was as follows:—The patient was thirteen years old on the 25th of last January. He has long suffered from glandular enlargements in the neck; but, with this exception, he had excellent health until about two years ago, when he was confined to bed for three weeks with severe pain in the back, between the shoulder-blades. He attributes this illness to an immersion which he had twelve months previously, in consequence of the ice giving way on the pond where he was sliding. He had no symptoms of any kind at the time, but is fully convinced that his illness, twelve months subsequently, was due to the wetting then received. The most careful inquiry failed to draw any account of this illness beyond the one fact of severe pain high up in the back. The pain did not extend to the arms; there was no loss of power, nor had he any febrile symptoms. In the course of six months he made a perfect recovery. About this time, or soon after, patient began to remark a curious trembling of the legs, which was excited if he sat upon a chair and touched the ground with his toes, but ceased when his heels were also pressed to the ground. He suffered little inconvenience from this symptom, and considered himself quite well until about last November, when he began to have pains in the left

elbow, and almost simultaneously with the onset of pain the left arm became weak, and he noticed that the muscles were beginning to waste. He thinks the pain, weakness, and wasting, were developed *pari passu*, and cannot assign any order to the advent of the symptoms. After a short time the same symptoms took place in the right arm. He was employed at the time in rope-works, and soon became unfit for his duties. He never had any tremors in the arms, nor any marked rigidity, though he thinks the muscles felt rather hard. He had some cramps and a feeling of stiffness in the fingers.

He gave up work in November owing to the weakness of the arms, and about this time his voice became rather feeble and husky, and has continued thus until the present. About the last week in January numbness began in the left foot, and a few days later in the right, and sensibility has continued impaired up to the present. About a week after the commencement of the numbness he became unable to walk owing to a rapidly-developing rigidity of the lower limbs, and was confined to bed until admission into hospital.

Present State.—The patient lies in bed, usually on the back or on the right side, and is unable to shift his position without assistance. He is well nourished, eats and sleeps well, is quite cheerful, and answers questions readily and with considerable intelligence. His legs are generally flexed on the thighs, and are so rigid that it requires considerable force to extend them, which the patient is usually quite unable to do of his own accord. More rarely the legs are found in a position of rigid extension, the patient being then generally quite unable to flex them. Occasionally he regains a very limited degree of flexion and extension, but soon loses it again. The muscles of the leg and thigh feel hard and tense to the touch, but are well developed, and show no trace of wasting. The condition of the muscles of the upper extremities is quite different. They feel soft and flexible, and there is marked wasting, and considerable loss of power. The rest of the muscular system presents no abnormality. The movements of the head and neck are perfect, and the patient retains complete command of the tongue, the eyeball, &c. Fibrillar twitchings may be frequently observed in the muscles of the legs. Sensation is notably affected. In the lower extremities tactile sensibility is greatly impaired, and at times almost lost, but it undoubtedly varies from day to day. The upper limit of diminished sensibility is not very easy to fix, the patient himself believing that he is affected from the umbilicus downwards. Tactile sensibility seems perfect in the upper portions of the body, including the arms. On the other hand, sensibility to heat is much increased in the legs. The patient informed me that for some time previous to admission he used to feel a scorching sensation in the legs when sitting before an ordinary fire, and on trying him with heated test-tubes the sensibility to heat was found to be very acute from the

thighs downwards. The sensibility to pain does not appear to be materially affected either by way of excess or deficiency. Electrical sensibility seems good in all parts, but the contraction of the muscles in response to the constant current is very defective in the legs, while remaining about normal in the upper extremities.

The patient suffers scarcely any pain; but when his legs remain long fixed in one position there is some aching on the anterior surface of the thigh if the limbs have been in the flexed, and on the upper surface of the foot if in the extended, position. As regards reflex action there is a marked difference between the superficial and deep reflexes. Of the former the plantar is the only one I have succeeded in obtaining after repeated trials, and even it is sometimes difficult to elicit. The cremasteric, gluteal, abdominal, epigastric, and scapular reflexes seem quite absent. The deep reflexes are present in a very marked degree. The patellar tendon reflex, or knee-jerk, is greatly exaggerated in both legs, but is sometimes difficult to obtain owing to the muscular rigidity. Ankle clonus can be very easily excited in both limbs, and is remarkably well developed. There has never been any bladder or rectal trouble, and the urine presents no abnormal features.

I have made a careful examination of the eyes, and the results of my observations have been confirmed and extended by the kind assistance of Dr. Nelson. The pupils are usually rather dilated, but respond readily both to light and to accommodation. There is at present some inequality between the pupils, which has only appeared since atropine was used for the purpose of ophthalmoscopic observation. The fundus in both eyes is rather pale, and the arteries are small relatively to the veins. There are one large and several smaller hæmorrhages visible in the left eye, and there are some chorioidal changes in each fundus, near the margin of the optic disc. The disc itself presents in both eyes some commencing atrophy, especially distinct at its outer margin, and faintly discernible at its inner edge. This atrophy is particularly important and interesting, as it does not occur in a case of pure lateral sclerosis, and, in conjunction with the other abnormal symptoms presented by this patient, may point to some implication of the posterior columns.

There can be no doubt that the symptoms in this case are mainly due to changes—probably sclerotic—in the lateral columns of the cord, but such an assumption does not definitely fix the diagnosis. Four hypotheses might be advanced—

1. That it is a case of primary lateral sclerosis.
2. That it is a case of lateral sclerosis secondary to some cerebral lesion.
3. That it is a case of lateral sclerosis secondary to some lesion of the cord.

4. That it is a case of multiple sclerosis mainly affecting the lateral columns.

The second hypothesis may be dismissed at once—the facts that the symptoms are bilateral, and that there is no history of any cerebral disease, being conclusive against it. The remaining three hypotheses cannot be so readily disposed of. Our knowledge of the differences between primary and secondary lateral sclerosis is not very exact, and multiple sclerosis is a disease which assumes such various forms that it is difficult to exclude it. On the whole, the evidence is against the probability of multiple sclerosis in this case. Some of the most striking features of that disease—such as tremor of all the affected parts on voluntary movement, nystagmus, syllabic articulation, vertigo, hebetude, &c.—are absent, or very slightly developed. The question thus narrows itself down to the issue of primary *versus* secondary lateral sclerosis, and in view of the history the latter seems the more probable. The severe and long-continued pain which the patient suffered from two years ago points to some disease of the cord taking origin about that time. What, then, was the probable nature and site of this disease? It must have finally involved the anterior cornua at the point of origin of the nerves going to the brachial plexus, as it was followed by wasting of the muscles of both arms, yet the history elicited bears but a slight resemblance to that of an attack of infantile paralysis. Again, are we to suppose that disease beginning in the anterior cornua in the cervical region was followed by sclerotic degeneration of the lateral columns in the dorsal portion of the cord? Charcot is of the opinion that where lateral sclerosis and degeneration of the anterior cornua coexist, the former is always primary, but in the present case the sequence of events would seem to be in the reverse order. The question hardly yet admits of dogmatic statement.

There is a form of lateral sclerosis—the *sclérose latérale amyotrophique* of Charcot—which begins in the cervical region, and spreads thence upwards to the medulla and downwards to the lumbar portion of the cord. It is characterised by paralysis, rigidity, and atrophy of the upper extremities, then by the development of a similar assemblage of symptoms in the lower extremities, and finally the medulla becomes involved, and we get paralysis and atrophy of the tongue and lips, followed by weakness of the muscles of the palate, pharynx, and larynx.

This is a form of sclerosis which we can hardly exclude with

certainly from the case before us. So far the decisively characteristic symptoms have not developed, though the feeble and husky voice of the patient might point to the implication of the medulla, and to the gradual onset of the phenomena which usher in the fatal issue of the amyotrophic form of lateral sclerosis.

I now propose to pass in review the most striking features of the case before us, endeavouring to explain them as fully as the present state of nerve pathology permits. I shall consider, firstly, the condition of the muscles as regards rigidity and atrophy; secondly, the anæsthesia; and, lastly, the very interesting group of reflex phenomena.

Rigidity.—Of all the symptoms in lateral sclerosis, none is more constant or better marked than the rigidity of the muscles. In the present case the rigidity of the muscles of the lower extremities is very extreme, but variable within narrow limits. Usually the legs are firmly flexed on the thighs, and the power of voluntary extension is almost entirely lost. It requires very considerable force to extend the legs, but nevertheless complete extension can be obtained, and then the patient is usually quite unable to flex the legs. Occasionally, however, he regains to some slight degree the power of flexion and extension, only to lose it speedily again. He retains some power over the hip-joint. What is the explanation of the rigidity in this case? It seems certain that it cannot be due to any changes in the muscles. No such changes have been described, and in the present instance the rapid onset of the rigidity, and the fact that it can be entirely overcome by force, are decisive against the existence of any permanent contractions in the muscles. The cause, then, is not peripheral, but central. If central, the further question arises whether it is spinal or cerebellar—the former being the usual view, the latter the opinion of so great an authority as Hughlings-Jackson. The former view is that the rigidity is of the nature of spasm, and is due to the hyper-activity of the reflex centres in the cord. In health these centres are held in check by cerebral influences transmitted to them along the path of the lateral columns, but in lateral sclerosis the cord is cut off from the inhibitory action of the brain, and so the reflex centres, being unbalanced, constantly exercise an unchecked predominance. The second view is that the rigidity is due to what Jackson calls “unbalanced cerebellar influx,” the influence of the cerebrum being cut off, and that of the cerebellum being proportionately augmented. In order to understand

this view, we must remember that in lateral sclerosis the degenerative process does not attack the whole transverse area of the columns. There is a zone to the outer margin of the lateral columns which escapes, and the fibres of this zone were believed by Flechsig and many subsequent observers to be connected with the cerebellum. But it is disputed whether these fibres are afferent or efferent in function. The former is the opinion of Bastian; the latter—adopted by Gowers—would harmonise with Jackson's view that along this tract the unbalanced impulses of the cerebellum pass to the muscles in cases where the cerebral influence is interrupted by sclerosis of the lateral columns.^a

Atrophy.—I do not propose to say much on this head, as the wasting of the muscles of the arms present in this case is not a characteristic sign, but only an accidental complication of lateral sclerosis. It is, no doubt, due to nutritive disturbance of the anterior cornua or the anterior roots, but it is mainly interesting to us at present as bearing on the question (to which I have already alluded) whether such degeneration, in cases where it is present with evidences of lateral sclerosis, is primary or secondary. The facts of the present case, so far as they go, tell rather against the view of Charcot that the disease in the anterior cornua is always secondary.

Anæsthesia.—There is marked loss of sensibility in this patient in the lower extremities, and though this is also an exceptional feature in lateral sclerosis, I propose to devote a little time to its consideration, as the testing of sensation is a matter of considerable delicacy, and requires not only much patience on the part of the observer, but some care and a fair amount of intelligence on the part of the patient.

^a It must be admitted that there is one fact which tells heavily against the view that this cerebellar zone is efferent. We know that in cases of transverse lesion of the cord an ascending degeneration travels up the cord, attacking mainly the posterior median columns, but also involving the cerebellar zone; and we know also that these secondary degenerations travel usually, if not universally, in the direction of the function of the parts involved. The fact therefore that the degeneration of the cerebellar zone spreads towards the brain is strong presumptive evidence that the function of this part is afferent and not efferent. Bramwell lays down the following points as diagnostic between primary and secondary lateral sclerosis. In the primary disease :—1. The onset is excessively chronic. 2. Motor weakness and rigidity are developed together. 3. There is no muscular atrophy at the upper level of the paralysis. 4. There are no sensory disturbances. 5. The bladder and rectum continue normal. On the other hand, in the secondary form the following points are to be noted :—1. The onset is much more rapid. 2. Motor weakness precedes the rigidity. 3. There is some muscular atrophy at the upper level of the paralysis. 4. Anæsthesia is usually present. 5. The bladder and rectum are frequently affected.

Sensation is a complex affair, and consists of, at least, three elements. There is sensibility to touch, sensibility to pain, and sensibility to temperature. These three activities are frequently affected in an entirely opposite manner—the diminution of one being often coincident with the exaltation of the others. Thus in anæsthesia the sensibility to touch is diminished or lost, while the sensibility to heat is frequently exalted. Again, it is not very uncommon to find the sensibility to touch perfect, while there is great impairment of the sensibility to pain—a condition to which the title of analgesia has been applied. Such facts as these, corroborated by the results of experimental research, have led to the belief that there are various paths for sensation in the cord, and the most recent view is that the sensation of pain travels by the gray matter—that of touch and temperature by the posterior columns. Recent observers assert that in some of the lower animals sensation travels partly by the lateral columns, but no facts have been adduced to prove that such is the case in man, and I am therefore disposed to regard the anæsthesia in the present case as not due to the disease of the lateral columns, but to the spread of the sclerosis to other portions of the cord. The case before us illustrates some of the anomalies of sensation to which I have alluded. The sensibility to touch is notably diminished—that to heat proportionately increased. I am rather doubtful as to the exact condition of the sensibility to pain, but am disposed to regard it as normal. In testing sensation there are several other points worthy of our attention. There is—

1. The quantity or degree of the sensation.
2. The quality or kind of the sensation—thus tactile sensibility may be perverted in various ways.
3. The localisation of sensation; and lastly—
4. The rapidity of transmission of the sensation.

To consider these points adequately would require more time than I can devote to them. Let me commend them to your attention, and more particularly the last point mentioned. There can be no doubt that mistakes often arise by sensation to touch or heat being pronounced defective, while it is really quite normal in amount, but simply delayed in transmission.

I turn next to the subject of reflexes—a group of phenomena which has already given us much aid in diagnosis, and which is probably destined still further to extend our knowledge of nervous diseases. Caution is, however, highly necessary in our deductions

from the various pathological abnormalities in reflex action, as there can be no doubt that in the enthusiasm of a new discovery many hasty conclusions have been drawn which subsequent observation has tended greatly to modify. Reflex action, like electricity, is capable of affording us valuable help, but it will fail, as electricity fails, if it is regarded as a sort of magic spell sufficient of itself to unlock all the mysteries of nervous disease.

We have to deal with two kinds of reflexes—the superficial or skin reflexes, and the deep or tendon reflexes—presuming for the moment that the latter phenomena are truly reflex in character, a presumption which is at least open to very considerable doubt. Of the skin reflexes the following are enumerated:—

1. The plantar reflex—viz., movements of the toes, or of the toes, foot, and leg, excited by tickling the sole of the feet.

2. The gluteal reflex—viz., contraction of the glutæi muscles, excited by irritation of the skin of the buttock.

3. The cremasteric reflex—viz., the drawing up of the testicle, excited by irritation of the skin of the upper and inner part of the thigh.

4. The abdominal reflex—viz., contraction of the abdominal muscles, excited by irritation of the skin of the abdomen.

5. The epigastric reflex—viz., a dimpling of one side of the epigastric region, excited by stroking the skin of the chest over the 5th and 6th intercostal spaces.

6. The scapular reflex—viz., contraction of the posterior axillary fold, excited by irritation of the skin in the interscapular region.

Of the so-called deep reflexes two claim our attention:—

1. The patellar tendon reflex or knee-jerk—viz., a single upward jerk of the leg and foot, excited by striking the patellar tendon with the edge of the hand or the percussion hammer whilst the leg hangs loosely over its fellow or over the forearm of the operator, also by striking the quadriceps muscle above the patella.

2. Ankle-clonus—viz., a series of clonic contractions at the ankle-joint, excited by extending or slightly flexing the knee, and then pressing quickly and firmly against the anterior part of the sole of the foot (so as to stretch the calf-muscles), and then keeping up the pressure.

I borrow the above tables from Charlton Bastian's article in Quain's "Dictionary of Medicine."

Is there any inter-relation or correspondence between these two

varieties of reflex activity? We shall find that there is no necessary relation, but that, on the contrary, either variety may be increased, diminished, or lost, without any corresponding change, or with, it may be, a change of an exactly opposite character in the other variety. In infantile paralysis both varieties are greatly diminished or quite lost. In some forms of paraplegia both varieties are markedly augmented. But as against such facts we have the following:—In many cases of hemiplegia we find the superficial reflexes lost, but the deep reflexes exaggerated. In posterior sclerosis or locomotor ataxia the absence of the deep reflexes is one of the most constant and important symptoms, but the superficial reflexes may be impaired or augmented, or may remain normal. In lateral sclerosis, on the other hand, the deep reflexes are greatly exaggerated, while the superficial reflexes may be almost entirely lost, as in the case before us.

From such facts one of three conclusions must be drawn—

1. That the superficial or the deep reflexes are not truly reflex in character; or,
2. That the path of sensory and motor impulses is different in the two cases;^a or,
3. That the brain exercises some influence in one case which it does not exercise in the other.

These suggestions open up a wide field for speculation, from which, however inviting it may be, I must at present turn away. Dismissing, then, the question of the differences between superficial and deep reflexes, while emphasising the clinical evidence of their independent variation, let us turn to the subject of reflex action in general and to the instruction which may be derived from the reflex abnormalities present in states of disease.

Reflex action is more vigorous in the young than in the old, in women than in men, in those who are called “nervous” than in the sluggish and phlegmatic. It is active during sleep, and possibly in all states of unconsciousness. It occurred to me recently to test the reflexes of a patient while under chloroform for the purpose of operation. I found the cremaster reflex well developed,

* This is the most probable explanation. Byrom Bramwell, in the admirably lucid and concise account which he gives of diseases of the cord in his work upon the subject, suggests that the sensory impressions for the superficial reflexes enter the gray matter directly by the posterior roots, and that those for the deep reflexes enter by way of the postero-external columns. The present paper was written before I had the advantage of consulting Bramwell's work. I find, however, that his account of lateral sclerosis confirms the view I have taken of the above case.

but the knee-jerk could not be obtained—a fact probably attributable to the circumstance that in this case there was only a very incomplete degree of muscular relaxation.

The information which the presence of a certain reflex gives us is very limited in its range, but yet most valuable. It shows us that the afferent impulse is safely conveyed from the peripheral point to the cord, that the reflex arc in the cord is intact, and that the path of the efferent impulse along the motor nerves is unimpeded. The various reflexes thus give us the means of testing the condition of the cord at almost any point—a fact which gives these phenomena their whole significance.

Let me indicate what the loss of reflex action teaches us; and I shall take as an illustration the patellar tendon reflex, because it has been very carefully studied, and we possess fairly complete and accurate knowledge regarding it. I feel justified in selecting this as a type, although its purely reflex character is open to doubt, because it is admitted by all that the lesions which prevent reflex action *pro tanto* prevent the development of the knee-jerk. It is evident, on theoretical grounds, that reflex action may be annulled by an impediment occurring anywhere in the reflex path. Practically we may confine ourselves to—1. Impediments to reflex action occurring in the course or distribution of the nerves involved; and, 2. Impediments occurring in the cord.

Thus, in pseudo-hypertrophic paralysis, the loss of the knee-jerk is probably due to conditions affecting the distribution of the nerves to the muscles. In experimental section of the nerves to the quadriceps muscle the loss of the knee-jerk is obviously the result of direct interruption of the nerve-currents.

The spinal causes of the loss of the reflex fall into three categories:—1. Cases where there is a lesion affecting the entire transverse area of the cord, as in some instances of paraplegia; 2. Cases of sclerosis of the posterior columns; 3. Cases of degeneration and atrophy of the anterior cornua.

As regards the first of these, it is easy to understand why there is an abolition of the reflex if the transverse lesion of the cord occurs at the point of entry of the afferent nerve involved; but Bastian is of opinion that in lesions of the cord in the cervical region there may be abolition of all the reflexes depending on the lower portions of the cord, though, as he adds, this view is contrary to the usual belief.

The loss of the knee-jerk in posterior sclerosis or locomotor

ataxia is apparently attributable to an interruption of the reflex arc on its sensory side. We know that the fibres of the posterior roots, before entering the gray matter, pass to a considerable extent through the postero-external columns, or posterior "root zone" of Charcot. It is this portion of the posterior columns which is exclusively affected in many cases of posterior sclerosis, and the loss of the reflex may be fairly explained by the bar to the passage of sensory impressions presented by the sclerotic degeneration.

Turning to infantile paralysis we find a different condition of affairs, the interruption to the reflex impulses occurring here in the motor portion of their path. The sensory impulse passes safely along the nerve, enters the gray matter of the cord possibly by way of the postero-external columns, traverses the fine network of fibres and cells comprising the central portion of the gray matter, and reaches the anterior cornua, where the existing disease prevents the origination of motor currents.

When we come to consider the conditions in which there is found an increase of the patellar tendon reflex, as in lateral sclerosis, hemiplegia, &c., we enter a more debatable and shadowy region in attempting any adequate explanation. The most generally received opinion is, that in all cases of an exaggerated knee-jerk there is sclerosis of the lateral columns interrupting the inhibitory influence which the cerebrum normally exerts over reflex action. That such an influence is exerted by the brain we can hardly doubt. We are all conscious that the will does exercise a restraining power over the reflexes. It cannot originate, neither can it entirely annul them, but it can alter and modify them in various degrees. So much is certain, but the increase of the knee-jerk might be due not merely to the withdrawal of the restraining influence of the cerebrum, but to the bringing into play of a new force—viz., that of the cerebellum. This view, to which I have made previous allusion, was originated by Hughlings-Jackson, and is at the lowest estimate a very fascinating speculation.

I shall next deal as briefly as possible with that phenomenon known as the ankle-clonus, to which we shall not be wrong in attributing great pathological significance.

I have already stated how it may be most certainly elicited; but in extreme cases, as in the patient before us, the clonus may be excited by attempts at voluntary movement, by a tap on the calf-

muscles, and even sometimes by tickling him over the sciatic nerve. The contractions occur at the rate of from six to ten per second. The clonus may be arrested sometimes (but certainly not always) by simple withdrawal of the pressure of the hand against the patient's toes, invariably by sharp flexion of the toes, but not—at least in the present case—by flexion of the great toe alone, according to the statement of Brown-Séquard. That the phenomenon is reflex in character seems probable; but Buzzard's view, that the recurrence of the contractions is explained by the pressure of the operator's hand causing a separate reflex act for each contraction, is disproved by the fact that in exceptional cases the clonus continues after the pressure is withdrawn. It may be that the sudden flexion of the toes which puts the tendo-Achillis on the stretch, sets up some rhythmical action in the nerve-centres; but such a speculation is of course only guess-work.

The ankle-clonus differs from the knee-jerk in the fact that the latter is present in health, while the clonus is essentially a symptom of disease. But when we come to inquire whether the latter is only found in cases of structural changes in the cord, we are confronted with a divergence of opinion. Gowers inclines to this view; but, on the other hand, Buzzard has found the clonus well developed in cases of hysteria where the subsequent recovery was perfect, in cases of paraplegia which ultimately disappeared; and Hughlings-Jackson has recorded a case of the clonus occurring in a patient immediately after an attack of unilateral convulsions. As regards the first of these points, it is always our duty to view with suspicion any argument drawn from any presumed hysterical phenomenon. The term hysteria, like the term struma, has been used to cover a multitude of sins—sins, that is, of diagnosis, and there is no doubt if we could eliminate those cases where there is structural disease and those where structural disease is complicated by hysterical phenomena, the residue would be small. Still the authority of Buzzard is too high to be lightly impugned, and I would therefore suggest, as a suitable subject for research, whether ankle-clonus ever occurs in cases of pure hysteria where we can eliminate with tolerable certainty the co-existence of organic nervous disease.

Buzzard's case of clonus in a paraplegic patient who recovered perfectly would, of course, be easily explained if we can admit there were structural changes in the cord which ultimately disappeared, and it is worthy of note that in this instance the clonus

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ceased to be developed with the amelioration of the general symptoms of the disease.

The instance recorded by Hughlings-Jackson of clonus occurring in a patient immediately after an attack of unilateral convulsions, is full of interest. His explanation is that the convulsions had caused temporary exhaustion of the lateral columns, which thus failed to transmit the inhibitory influence of the cerebrum, but it would be hard to prove certainly that the patient had no cord disease.

It is a matter alike of interest and importance to determine the exact clinical value of the ankle-clonus. At present I think we may safely conclude that if it is well developed, and persists for a considerable time, there is probably organic disease of the cord. If it be slight, we should perhaps wait until we see if it persists before basing a positive opinion upon it.

A very interesting point connected with the ankle-clonus was brought forward at a recent meeting of the Edinburgh Medico-Chirurgical Society—viz., that the rapidity of the clonus was in inverse ratio to the height of the individual—a fact that was regarded as strongly corroborating the view of its reflex character.

As regards the treatment and progress of this case a few words will suffice. He was treated first with the iodide, and then with the bichloride of mercury, but any occasional amelioration of his condition proved of transient duration, and the prognosis in this, as in most, if not all, cases of lateral sclerosis, leaves little hope for anything but a steady, if slow, aggravation of his symptoms until death shall close the scene.

ART. III.—*On the Nature of the Phosphatic Precipitate obtained upon Heating Urine.* By WALTER G. SMITH, M.D.; King's Professor of Materia Medica, School of Physic, Trin. Coll. Dubl.; Physician to Sir Patrick Dun's Hospital.^a

THE fact that clear urines, free from albumen, and neutral or even faintly acid in reaction,^b frequently become turbid upon heating is familiar to every one. The turbidity readily and completely disappears upon the addition of a few drops of dilute acid;

^a Read before the Dublin Biological Club, April 24th, 1883.

^b Acidity of healthy normal urine is probably due (a) to acid phosphate of Na; (b) acid urate of Na; and (c) ? to free aromatic acids.